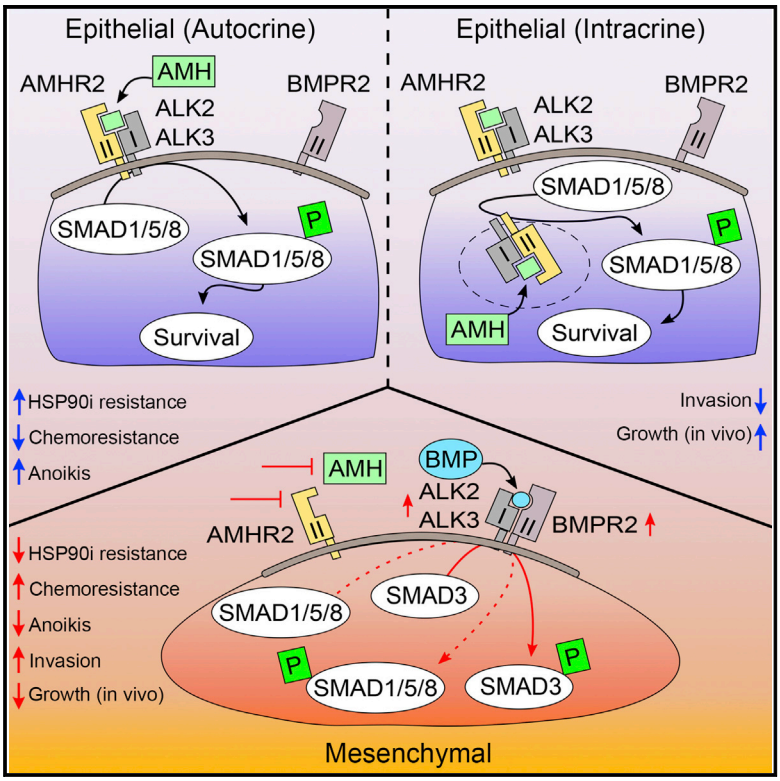
Article

Cell Reports

Anti-Müllerian Hormone Signaling Regulates Epithelial Plasticity and Chemoresistance in Lung Cancer

Graphical Abstract



Highlights

- TGF-β superfamily member AMH regulates tumor growth and drug resistance in NSCLC
- AMH and AMHR2 activity influences SMAD, AKT, and NF-κB signaling in NSCLC cells
- Loss of AMH/AMHR2 promotes EMT through direct modulation of TGF-β/BMP receptors
- EMT promotes chemoresistance, but sensitizes NSCLC cells to HSP90 inhibition

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In Brief

Beck et al. identify active signaling by the TGF-β/BMP superfamily member anti-Müllerian hormone (AMH) and its receptor AMHR2 in non-small cell lung cancer (NSCLC), demonstrating a role for AMH/AMHR2 in influencing the basal and BMP-dependent SMAD signaling that constrains epithelial-mesenchymal transition (EMT) and in regulating drug resistance.

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